Relationships among diseases and other small-scale disturbance processes, forest growth and development, fuel dynamics, and wildfire spread and behavior: A review of the literature

Final Report

Relationships among diseases and other small-scale disturbance processes, forest growth and development, fuel dynamics, and wildfire spread and behavior: A review of the literature

Ann E. Camp, John E. Lundquist, Kelly M. Droege, and Jay McLaughlin

Abstract

Introduction

Vegetation patterns across landscapes arise from the interaction of forest development with biotic and abiotic disturbances. These vegetation patterns determine the wildlife habitat value of different forested landscapes. An extensive body of literature addresses relationships between vegetation patterns and wildlife use (Harris, 1984, Hunter 1990, Oliver et al. 1997). A newer focus on forest vegetation patterns is emerging as large landscapes, especially those in the inland Western United States, are consumed each year by catastrophic wildfires - fires many believe are unprecedented in size and severity. In many instances, fire exclusion during the past century is blamed for increasing fire severity (Covington et al. 1994). Absent frequent but low severity fires, forests have developed further along successional trajectories. Throughout this process of forest development, pathogens, insects, and other small-scale disturbances kill individual and groups of trees, influencing amounts and distributions of woody debris (Maser et al. 1979) that is helping fuel fires across entire landscapes in the interior Western United States.

The purpose of this paper is to review existing literature to determine and synthesize existing knowledge about the interactions between forest developmental stages and small-scale disturbances (primarily diseases and disease complexes) within the interior western United States. Information obtained from reviewing the literature will presented, to the extent possible, in terms of potential quantities, qualities and expected spatial and temporal patterns of fuels. Knowledge of the relationships between forest developmental phases

and the accumulation of fuels through tree mortality will be important for defining landscape patterns that are sustainable under inherent fire regimes and prescriptive with respect to minimizing fire risk.

Forest development and corresponding fuel levels

Forest stands and thus forested landscapes are not static; forests develop along a continuum during which trees establish and grow, compete with each other for light, moisture, and nutrients, and eventually decline and succumb to biotic and abiotic disturbances. The rates at which individual forest stands develop give rise to a changing kaleidoscope of vegetation patterns across landscapes. Oliver (1981) and Oliver and Larson (1996) proposed and characterized four phases of stand development which they term stand initiation, stem exclusion, understory re-initiation, and old growth. Others have somewhat differently named and divided the continuum of forest development (Bormann and Likens 1979, O'Hara et al. 1996, Franklin et al. 2002). What each nomenclature has in common, however, are developmental phases dominated by regeneration, competition among individuals for site resources, gap formation and patch dynamics, or some combination of these processes. In this paper, the terminology used by Oliver and Larson (stand initiation, stem exclusion, understory re-initiation, and old growth) will predominate; however other terminology will be used occasionally to highlight specific points. 'Forest development' is similar to forest succession in that both terms refer to changes in species composition, structure, and function over time, however forest

development does not infer the orderly progression to some fixed endpoint that is sometimes implied by succession.

Throughout forest development, some trees die. While death of trees occurs during all phases of forest development, the amount, pattern, and characteristics of this mortality differ. Dead trees- snags and down logs -are simultaneously an important component of wildlife habitat and potential fuel for wildlfires. Very little exists in the literature that specifically ties small-scale disturbances to phases of forest development (but see Lewis and Lindgren, 2000 for a discussion of this with respect to the central interior of British Columbia). It cannot be argued, however, that mortality patterns and fuel accumulation change over time; some of these changes can be inferred from the literature.

The start or initiation of a forest is generally precipitated by the wholesale death of the previous forest from some disturbance. Disturbances that include timber harvest or salvage may remove most or all the larger dead tree component (tree boles) from the site. Other disturbances produce regenerating forests that include many residual snags and logs. Fall and decay rates of these 'biological legacies' (sensu Franklin et al. 2002) are a function of mortality agent, climate, and size and species of the dead trees (Dahms 1949, Mielke 1950, Keen 1955, Kimmey 1955, Lyon 1977, Bull 1983, Schmid et al. 1985, Harmon et al.1986, Parks and Shaw 1996, Bull et al.1997). From a fuels and fire behavior perspective, the stand initiation phase of forest development can begin with a wide variation in the amount and type of fuel.

Fuel levels through the stand initiation phase and beyond is a function of decomposition rates and recruitment of additional fuel through the death of residual live trees in the regenerating stand. There may be extensive mortality within the cohort of seedlings establishing after a disturbance (Fowells and Stark 1971, Tappeiner and Helms 1971); however, the amount of biomass generated is relatively low and decomposition rates probably high owing to the small size of the material

Existing literature about dead trees in regenerating forests focuses almost exclusively on their value to wildlife (Thomas et al. 1979, Maser et al. 1979, Bull et al. 1997) or in providing soil organic matter and other functions relating to site productivity (Harmon et al. 1986, Jurgensen et al. 1997). Franklin et al. (2002) explicitly discuss amounts and conditions of biological legacies arising from various stand initiating disturbances and the necessity for incorporating dead trees in managed forests to more closely mimic 'natural' stands; their paper does not address residual mortality from a fuels perspective. ,

Once a forest has successfully regenerated, it enters a developmental phase characterized by competition among trees for limited resources, resulting in the death of less competitive individuals. Variously termed the Aggredation (Bormann and Likens 1979), Stem Exclusion (Oliver 1981) or Biomass Accumulation / Competitive Exclusion (Franklin et al. 2002) phase, this period of forest development accounts for the greatest amount of postestablishment tree mortality (Peet and Christensen 1987). Trees die when they are at a competitive disadvantage because of such factors as size, vigor, and proximity to

neighboring trees. Mortality is linked to the relative size of trees within the stand, with smaller trees more likely to die than larger ones. Over time, total stand and average individual tree biomass increases as the number of trees decrease. An entire body of mensurational literature exists describing and quantifying this stage of forest development, predominantly for forests of a single age class and single species (i.e. plantations). Yoda et al. (1963) were the first to quantify the tendency for competing vegetation to self-thin, however the predictive capability of the self-thinning or -3/2 rule is limited to stands with a single age class and single species.

Although competition is the underlying cause of mortality during the thinning phase of development, the immediate agent of death is generally from an insect or pathogen that opportunistically attacks and kills the weakened tree. As competition-driven mortality progresses, the pattern of living trees tends to shift from clumped to random to regular (Curtis and McIntosh 1951, Cooper 1961, Laessle 1965). The pattern of fuels created by tree mortality can thus also be expected to shift as development progresses throughout this phase. The amount and size of fuels also increases throughout this phase of development.

As forests mature, trees grow in height and girth; death of individual trees create larger openings in the forest canopy and forest development moves into phases characterized by the formation of canopy gaps or openings that are large enough to support the establishment of new cohorts (age classes) of trees. Of course, at any point along the developmental continuum, a large-scale disturbance can - and often does - reset the biological clock back to a

regeneration-dominated phase. Tree death during the understory re-initiation and old growth phases of stand development (terminology after Oliver 1981) is density independent, with diseases, insects, lightning, and windthrow killing individual or small groups of trees. These disturbances can be chronic, resulting in a relatively constant level of mortality per year, or episodic, killing many trees at less frequent intervals. Different tree species have different expected life spans. In a mature forest, some species may be expected to live for many decades or even centuries barring outside disturbances while other species may be nearing their maximum expected age; thus, species composition is another factor influencing the expected level of mortality for a particular forest.

Based on forest development theory, mortality and total fuel amounts will be highest at the onset of the regeneration phase and continue to build throughout the competition phase. Fuel characteristics will differ over time, with fuels in all size classes present at the onset of regeneration (unless the larger portion is removed through harvesting), small and large fuels accruing during regeneration and progressively larger fuels accumulating during the competition phase. In later phases of forest development, the largest fuel classes will again be recruited. Rates and persistence of fuel accumulation are a function of stand density and decay efficiency. Forests in hot, dry environments and those growing in colder areas will tend to accumulate fuels since both environmental conditions retard decay processes.

Forest development and small-scale disturbances

Introduction

Small scale disturbances that create structure within forests and contribute to the accumulation of fuels can be biotic (pathogens, insects, human management) or abiotic (lightning, wind, ice). Not surprisingly, the literature on disturbances in the inland West is heavily weighted toward the effects of wildfire and large-scale biotic disturbances such as outbreaks of defoliating insects and bark beetles. Fire is discussed as a primary disturbance agent in the inland West and fire exclusion as a driver of stand and landscape level changes in forest development patterns. Insects and diseases are frequently characterized as inefficiently substituting for fire in regulating forest ecosystem function. Their presence is often cited as evidence for ecosystem dysfunction related to past fire suppression policies (Hessburg et al. 1994 Steele 1994). With some notable exceptions, native insects and diseases are the primary non-human biotic disturbance agents in interior Western forests. The exceptions, however, have increased mortality in host species over historic levels; these introduced or exotic agents include white pine blister rust, larch casebearer, and balsam woolly adelgid (Mitchell 1966).

Pathology and entomology literature identifies specific forest conditions and environmental factors promoting or retarding insect and pathogen activity. Like other species of wildlife, insects and pathogens are more successful in some forest conditions and less so in others. Differences in tree density, species composition, canopy structure, and landscape-scale forest vegetation patterns will favor some insects and pathogens over others (Hessburg et al. 1994).

Root and stem decay pathogens

The three primary interior western root diseases are laminated root rot caused by *Phellinus weirii* , Armillaria root disease, caused by *Armillaria ostoyae* and other *Armillaria* species, and P and S type annosus root disease caused by *Heterobasidion annosum* (Goheen and Filip 1980, Hadfield et al. 1986). Smith (1984) estimated a mean annual volume loss to five major root diseases (the three previously mentioned plus Phaeolus schweinitzii and Ophiostoma wageneri) in the western United States of 6.7 million m^3 or 18% of the total tree mortality. Shaw et al. (1976) found that per ha volume loss to Armillaria in a ponderosa pine stand in eastern Washington increased from 9 m^3 in 1957 to 24 m^3 in 1971. Filip (1977) estimated that 32% of the standing volume (but only 7% of the trees) in a mixed conifer stand in southern Oregon was infected with or had been killed by Armillaria, while Filip and Goheen (1982, 1984) reported annual mortality from Armillaria at > 3 m^3 per ha.

Susceptibility to root diseases differs among host tree species and location, with grand fir and Douglas-fir being very susceptible to Armillaria, annosus root disease, and laminated root rot throughout most of the inland West (Morrison 1981, Hagle et al 1987, Hagle and Goheen 1988). In south-central Washington, ponderosa pine is very susceptible to Armillaria, while Engelmann spruce and western hemlock are extremely susceptible to root pathogens in parts of their ranges (Morrison 1981, McDonald et al. 1987b). Susceptibility also changes with age; in conifers, incidence of mortality from Armillaria generally

decreases with age as host resistance to infection increases (Buckland 1953, Johnson et al. 1972). That relationship is reversed for annosus root disease and laminated root rot, with older trees becoming increasingly susceptible.

The age at which western conifers become more resistant to Armillaria infection tends to occur sometime during the stand initiation or early stem exclusion phases of forest development. As Armillaria infection is more likely in stressed host trees, (Davidson and Rishbeth 1988) it probably plays a role in killing trees weakened by competitive interactions during the stem exclusion phases of development where it functions as an opportunistic or secondary pathogen. In some forests, especially those dominated by Douglas-fir and true firs, Armillaria-induced mortality can occur within a few years of regeneration and continue throughout forest development. In Armillaria-infected forests dominated by western redcedar, western larch, mountain hemlock, or pine, mortality attributed to Armillaria decreases after about 30 years (Kile et al. 1991).

Russell et al. (1973) found significant regional differences in annosus root disease infection following pre-commercial thinning of ponderosa pine in eastern Washington, with rainfall amounts explaining much of these differences. Their results highlight some inherent problems with using forest development as a predictor of mortality associated with small scale disturbances: first, management activities can strongly promote or retard susceptibility, and second, factors unrelated to forest development are often significant variables with respect to susceptibility and/or mortality. This study did, however, indicate that susceptibility to annosus root disease is greater in managed mature forests than in managed

young (stem exclusion phase) forests. Variables responsible for observed differences included tree size, age and site moisture. Annosus root disease spreads via innoculation of cut stumps; partial harvesting in mature stands provides a larger 'target' of cut stumps than does pre-commercially thinning young stands, thus increasing susceptibility. Differences in moisture regime between the two areas studied (the mature forests were in California and the young forests in eastern Washington) may also have contributed to the results observed.

Mallett and Maynard (1997) found that the sand content of soil was significantly correlated with presence of *Armillaria* in young lodgepole pine stands in Alberta, BC whereas age and stand density were not. McDonald et al. (1987*a.b.*) concluded that the highest incidence of *Armillaria* in unmanaged subalpine fir and Douglas-fir forests occurred in the transition zones between cold-dry and cool-moist regions and between hot-dry and warm-moist regions. Cold dry and hot dry sites had the lowest incidence of *Armillaria*. From a fuels perspective, lower levels of mortality might be offset by longer decay rates such that fuels on these extreme sites might persist longer than on mesic sites having higher mortality.

In a seminal paper addressing the role of pathogens on forest patterns and processes, Castello et al. (1995) suggested that slow spreading pathogens such as dwarf mistletoe and root and butt rot fungi, function primarily as gap makers in older forests. The root pathogen P. weirii appears to function in this manner in subalpine forests in Oregon; the pathogen only becoming noticeably

apparent after 200 years (Dickman and Cook 1989). Research by Byler et al. (1990) also support the notion of slowly spreading pathogenic fungi functioning as gap makers in old forests. Douglas-fir dominated forests in Montana with root disease patches were older than similar forests having only scattered mortality from pathogenic fungi. Scattered mortality in younger stands is likely attributable to isolated, opportunistic pathogens delivering a coupe d' grace to individual trees that have been outcompeted by neighboring trees. In contrast, disease patches may gradually spread outward, killing otherwise healthy trees of susceptible species. Species most susceptible to root pathogens are often shade-tolerant and can establish and grow in forest gaps, thus helping perpetuate fungal presence in older forests. Filip and Goheen (1984) reported severe mortality (up to 55 percent of the trees and 39 percent of the basal area) from *Phellinus weirii*, *Armillaria mellea* (sic), and *Heterobasidion annosus* in uneven-aged stands dominated by white and grand in eastern Washington and Oregon.

In a study designed to relate the presence of a suite of root and stem decay pathogens to stand composition and elevation, Hobbs and Partridge (1979) were unable to correlate the incidence of decay to either tree or stand age. They attribute this lack of correlation to a small sample size (74 stands) and the fact that many forest stands in their sample were uneven-aged. They noted that the stem decay fungus Ecinodontium tinctorum occurred most frequently in forests where grand fir dominated or shared dominance; infected grand fir were either "overmature" or "occupying subordinate positions within the canopy." They further found that Phellinus pini, another stem decay pathogen was most

frequently found in forests dominated by western larch, with increased frequency below 1500 m. Forest composition could not explain distributional patterns of three other pathogens: Phaeolus schweinitzii, Inonotus tomentosus, or Perenniporia subacida.

A study specifically tying pathogen activity to forest conditions, (though not developmental phase *per se*) was reported by Hagle (1985) from research conducted in the Idaho panhandle. Root disease severity was higher in grand fir and western hemlock series forests compared to those in the Douglas-fir series, and significantly related to aspect, with higher severity in forests on northeastern, eastern, and southeastern aspects. Species composition - relative proportions of Douglas-fir and grand fir - influenced mortality levels, with Douglas-fir having twice the mortality (4.1%) as grand fir (2.2%). No mechanism for the relationship with aspect was hypothesized, nor was pathogen severity found to be related to slope steepness or elevation.

As forests mature and become dominated by shade tolerant species, virulent root pathogens are better able to spread along the interlacing and connected roots of host species. Based on research undertaken on Vancouver Island, Reynolds (1980) argued that tree, stand, and site factors that increase the probability of inter-tree root contact are the dominant variables influencing Phillinus weirii hazard and that root contact potential can be used as a direct measure of spread potential. Root contact probability increases with conditions that favor development of larger trees with extensive root systems and with conditions that decrease species diversity. Large trees are found in older forests,

in young forests with residual trees that survived the initiating disturbance, and in forests that have been heavily thinned by natural or mechanical means. Conditions favoring development of shade tolerant understories can result in increased root contact between susceptible host species.

Hinds (1977) discusses relationships between decay, tree diameter, and age for Engelmann spruce and subalpine fir in the Rocky Mountains. Losses from root and stem decay range from less than 7% (based on board feet) for trees under 10" dbh to over 40% in trees over 20" dbh. Decay was infrequently present in trees < 100 years old but rapidly increased with age. From 150 - 199 years, the amount of decay was about 35%. Camp et al. (1997) found decay incidence in true firs also increased dramatically in individuals > 100 years.

Lewis and Lindgren (2000) found that the abundance of various decay fungi and other biotic disturbance agents varied as stands progressed through developmental stages. While they conceded that most root diseases are generalists with respect to age of host trees, older trees with larger root systems were more likely to form root contacts with inoculum sources. In contrast, stem diseases caused by canker or rust fungi were more apt to kill young trees with small diameters since less time was required to girdle them. As forests age, mortality often accrues on groups of similar trees in close proximity and is thus distributed less randomly and may be strongly correlated with underlying physiographic, edaphic, or topographic conditions (Byler et al 1990).

Heart-rot fungi are spread by airborne spores and colonize freshly exposed wounds or branch stubs. Spores of heart rot fungi are abundant in all forests, but old-growth forests have a much greater incidence of heart rot than younger forests (Manion 1981). Heart rot fungi are less pathogenic than many root disease fungi, allowing infected trees to persist for decades (Bull et al. 1997).

Many of the papers reviewed above note that root decay incidence and mortality are greater where trees are stressed by environmental conditions. Moore et al. (2000) relates this to lower concentrations of carbon in the roots of trees growing on moisture limited sites. Armillaria in particular appears able to exploit this lowered resistance. Mortality from root decay fungi occurs in young and older forests; in younger forests these agents generally act as scavengers, killing smaller trees weakened by competition. Dead trees (fuels) will occur in a scattered or random pattern. In older forests, virulent root decay pathogens often form disease centers; mortality will be clumped, with clump sizes increasing over time. Forests with a few, small root disease centers will accrue less fuel than those with many, large disease centers. Highest accrual and greatest fire risk will be in forests where individual disease centers coalesce.

Dwarf mistletoes

Dwarf mistletoes (Arceuthobium spp.) are a group of parasitic higher plants confined to conifers. Most of the known species occur in western North America and cause extensive growth loss and mortality, especially in ponderosa

pine, Douglas-fir, and lodgepole pine. In some areas western larch is also severely impacted. Mortality levels vary considerably, depending on the host/parasite combination, stand age, and site factors (Hawksworth 1978); in addition to directly killing trees, dwarf mistletoe infections reduce tree vigor and increase vulnerability to secondary pathogens and insects. Dwarf mistletoes may be especially important contributors to tree mortality during periods of drought or when stand density is high (Byler 1978).

Dwarf mistletoe infections spread slowly, with the most rapid spread being between proximal overstory and understory trees. The relationship between dwarf mistletoe infection and multi-canopied forests is well-documented (Gill and Hawksworth 1954, Graham and Frazier 1962, Scharpf and Hawksworth 1968, Shea and Stewart 1972). Young trees growing beneath an infected overstory of the same species are at high risk of being infected. Because there is a high degree of host specificity with the various species of Arceuthobium, mixed species forests are less vulnerable than monocultures.

Bolsinger (1978) found dwarf mistletoe incidence increased with tree size in forests dominated by hemlock, western larch, true fir, or Douglas-fir; no such trend was found for lodgepole pine, and a weakly reverse trend was observed for ponderosa pine. Incidence of a particular species of dwarf mistletoe may (Alexander 1975, Hadfield 1977, Merrill et al. 1987) or may not (Childs and Edgren 1967, Parmeter 1978) be related to site quality; however impacts on the growth and mortality of the host is strongly related to site quality (van der Kamp 1987, Hawksworth and Johnson 1989).

Historically, fire played a primary role in the distribution and abundance of dwarf mistletoe (Alexander and Hawksworth 1975). High severity fires effectively halted infestations while less severe fires perpetuated dwarf mistletoe infections by leaving infected residual trees or maintaining seral forests composed of susceptible hosts (Hawksworth and Weins 1996). Fire exclusion in the Rocky Mountains allowed less susceptible spruce and fir forests to replace lodgepole pine, a species highly susceptible to Arceuthobium americanum. Forests with dwarf mistletoe infestations accumulate an abundance of fine fuels and ladder fuels that can result in crowning fires.

Trees are susceptible to dwarf mistletoe infestation at any age but very young or very small trees are rarely infected because their small crowns make poor targets for disseminating seeds (Parmeter 1978). Many studies confirm that the number of trees infected, infection severity, and amount of damage increases with tree age (Hawksworth 1960a, Baranyay 1970, Stewart 1976, Hadfield 1977). Mortality rates are generally high in old stands that are heavily infected with mistletoe, although mistletoe plants may produce less seed on old trees (Roth 1953).

Mistletoe-caused mortality is difficult to quantify. Trees infected by mistletoe lose vigor and may be attacked and killed by insects. Most reported mortality comes from U.S. Department of Agriculture, Forest Service literature; use therein of regional board feet metrics to report mortality disguises stand-level mortality rates. Andrews and Daniels (1960) estimated annual mortality of ponderosa pine in the southwestern US at between 55-75 million board feet. It is

unlikely that mortality was uniform throughout the region and these estimates preclude assigning mortality levels to different forest developmental phases; however, much of the mortality probably accrued within older forests and forests having few species.

Other pathogens

Other pathogens that kill trees in the inland West include those causing loss of foliage and those that girdle stems. The latter include canker fungi such as Atropellis piniphila that primarily attacks lodgepole pine, comandra blister rust that attacks and sometimes kill lodgepole and ponderosa pines, and white pine blister rust, an introduced pathogen that kills 5 needle pines. The impact of hard pine stem rusts (western gall rust, stalactiform blister rust, comandra blister rust) occurs mostly in young forests (stand initiation phase of development) since the more susceptible lower branches rapidly die in the reduced light environment following crown closure. Reduced light availability at the forest floor during the stem exclusion phase of forest development can also limit populations of the rust pathogens' required alternate host species. Unlike many pathogens, stem rust impacts are greatest on vigorous trees; conditions promoting tree growth also promote rust growth. Because of this and the tendency for their aggregated distribution within forests, stem rusts are generally not particularly effective thinning agents.

While damage from canker causing pathogens can be lethal, trees weakened by canker pathogens often succumb to root diseases and bark beetle

attacks. Comandra blister rust attacks lodgepole and ponderosa pine of all ages and sizes, but seedlings are especially vulnerable and generally die within a few years. Direct mortality in older trees takes about twice as many years as the diameter of the bole where the canker occurs; mature and old trees often exhibit dead tops that can persist for many years (Johnson 1986). Comandra blister rust generally operates at endemic levels; when localized outbreaks occur, they are associated with weather events rather than stand conditions.

White pine blister rust caused waves of mortality in western white pine after the disease was accidentally introduces from Europe at the beginning of the 20th century. Currently the disease prevents most young western white pines from reaching maturity.

Needle cast diseases negatively impact tree growth but are usually not lethal unless they occur several years in succession. While trees of all ages are susceptible, mortality from needle cast pathogens primarily occurs among seedlings and saplings. Needle cast disease occurrence more related to site and weather conditions than forest developmental phase.

Elytroderma deformans is a pathogen that causes needle loss in ponderosa pine. On plots where Elytroderma was present, mortality from other agents ranged from 0.25% to 2%, with increasing severity of Elytroderma infection and decreasing tree vigor measured by Keen crown class (Childs 1968). Thickets of reproduction can be heavily infected; infection intensity is highest under conditions of moderate overstory density. Trees weakened by this disease are often attacked by bark beetles or killed by root decay fungi. Direct mortality

from Elytroderma occurs after 2-3 years of severe defoliation and generally operates at a scale of up to about 100 acres. (Childs et al. 1971.

Insects

Insects in inland western forests operate at various spatial scales, from individual trees to landscape-scale outbreaks. These latter episodes are outside the scope of this paper; however, the point at which endemic levels of insect activity become outbreaks has not been clearly defined. Mortality from insects is a function of forest condition (including developmental phase) and environmental conditions that sustain insect populations (Safranyik 1985). As forests develop, their associated insect populations change (Schowalter et al. 1997, Lewis and Lindgren 2000). Trees in young forests are particularly susceptible to weevils and other insects that affect tree form, but do not cause extensive mortality. As forests develop, trees become increasingly hospitable to defoliators and bark beetles, both of which can result in extensive mortality at varying spatial scales. Non-lethal defoliation can reduce vigor to the extent that trees become increasingly susceptible to mortality from other biotic agents, including bark beetles and pathogens. Among native interior western insects having the potential of causing high levels of mortality are bark beetles (Dendroctonus, Solytus, and Ips species), western spruce budworm, western hemlock looper, and Douglas-fir tussock moth. Effects of these insects are partly a function of stand condition and partly caused by environmental conditions, especially droughts.

Susceptibility of forests to defoliation by western spruce budworm is a function of species composition, regional and site climate, stand density and structure, tree vigor, maturity, and the surrounding landscape matrix (Carlson and Wulf 1989). Marsden et al. (1986) determined that severe top kill and mortality from western spruce budworm on the Payette National Forest in Idaho was greatest on grand fir and lower elevation Engelmann spruce/subalpine fir habitat types. Susceptibility also increased with increasing stand density and maturity. Other researchers have found that the most susceptible stands had some or all of the following characteristics (Marsden et al. 1986, Wulf and Cates 1987): they occured in areas of low winter temperatures; were sited on droughty soils, and often on steep slopes; contained a high percentage of shade tolerant tree species; had a high density of low vigor trees; contained trees that were mature or old; had a multicanopy structure; and were surrounded by or downwind from infested stands.

Bark beetles

In summary, mortality from insects tends to increase with increasing age in inland Western forests. Competitive interactions during the stem exclusion phase of development weaken some trees that may succumb to bark beetles operating at endemic levels. A pulse of dead trees may allow bark beetle populations to grow to the point where living trees are attacked; but unless those live trees are big enough to support reproduction requirements, beetle populations will again fall. Defoliators, too, fare better in older forests because

species compositions and structures are better suited to larval growth and dispersal.

Interactions among multiple stressors

Information about mortality levels arising from biotic agent complexes is very limited. Numerous researchers have reported an exacerbating impact of bark beetles in root disease centers (Lessard et al. 1985, Schowalter and Filip 1993, Holah 1994). Other scientists have explored relationships between a biotic disturbance agent and an abiotic one, such as dwarf mistletoes and fire or lightning and bark beetles (Rykiel et al. 19xx). And still others have postulated interactions initiated by an agent or condition that enhances tree susceptibility to an agent that inflicts mortality (Sinclair 1967, Manion 1981, Wargo or Houston 19xx, Lundquist 1993). Taken together, this body of literature points to increased levels of mortality when more than one agent is involved.

Multiple root pathogens often occur together in inland Western forests (Hobbs and Partridge 1979, Goheen and Filip 1980, Filip and Goheen 1984, Hansen and Goheen 1989), colonizing roots of adjacent trees or even roots of the same tree. Some evidence suggests that coincidence of multiple root pathogens are indicative of successional relationships among various types of fungi. For instance, Goheen and Hansen (1978) found that *Leptographium wageneri*, the causal organism of black stain root disease, appeared to predispose ponderosa pine, mountain hemlock, and Douglas-fir to *Armillaria*.

Elevated mortality is also associated with interactions between root disease pathogens and dwarf mistletoes. Byler (1978) found such associations accounted for 11-28% of conifer mortality on four National Forests in California. Root diseases and bark beetles often co-occur with Cronartium ribicola, the introduced pathogen that causes blister rust in North American white pines (Kulhavy et al. 1984, Camp unpublished data).

Forest management can be a precursor to increased mortality from biotic disturbance agents. While thinning should increase the vigor of residual trees by releasing them from competition, several researchers have documented increased mortality from Armillaria root disease following partial harvests (Filip 1977, Redfern 1978, Filip and Goheen 1982). Wargo and Harrington (1991) suggest that site conditions, age of the thinned stand, pre-harvest condition of trees in the thinned stand, and pathogenicity of the Armillaria species will determine how partial cutting affects Armillaria root disease and subsequent Armillaria-induced mortality. Lewis and Hansen (1991) demonstrated that forest management practices that accelerate early stages of forest development in sub boreal forests in British Columbia subsequently resulted in increased mortality from the root pathogen Inonotus tomentosus.

Mortality from both primary and secondary bark beetles increases following disturbances that produce large amounts of dead and dying trees. Windthrow of spruce and Douglas-fir are associated with elevated mortality from spruce beetles and Douglas-fir beetles, respectively (Safranyik 1985). Woody debris left after harvesting can increase Ips pini populations (xxx), causing this

generally secondary bark beetle to become a killer of living trees. Lane and Goheen (1979) and Filip and Goheen (1984) reported that in their studies of root diseased white and grand fir in eastern WA and OR, they frequently encountered heavy infestations of bark beetles, especially fir engravers (Scolytus ventralis).

Cumulative effects on tree mortality from interactions among multiple disturbance agents operating at small scales are likely to increase through time and will thus manifest increasingly within older forests.

Fuel quantities and qualities

While large insect outbreaks are acknowledged to increase the horizontal continuity of fuels and dwarf mistletoe infections have been linked to formation of fuel ladders, very little quantitative information exists about the relationship between small-scale disturbances and their effects on quantities and qualities of fuels. Reports on mortality associated with such disturbances indicate that the relationship does exist and may significantly impact fuel quantities and patterns across inland Western landscapes. James et al (1984) found that active root disease centers occupied nearly 32,000 ha or 1% of the total commercial forest land on national forests in the northern Rocky Mountains. Dickman and Cook (1989) reported high levels of mortality from Phellinus may affect the initiation and spread of fire in high elevation forests dominated by mountain hemlock. Filip and Goheen reported mortality of between 34 and 133 stems per ha from Armillaria, Phellinus, and Annosus in 14 heavily infected white and grand fir stands and estimated an annual mortality rate of 0.1% of the total white and grand fir volume, or 183,200 m^3. Stands incurring the greatest amount of mortality included those that had been partially harvested; they postulated that cutting large-diameter living infected trees could increase inoculum potential and thus mortality from root diseases (Filip 1977, Filip and Goheen 1982).

Koonce and Roth (1985) determined that mistletoe-infested stands of ponderosa pine had higher total fuel loads; branches infected by dwarf mistletoe were larger, more resinous, and persisted longer than healthy branches. Many

infected trees have highly flammable witches brooms and lower live crowns, allowing ground fires to move into crowns (Harrington and Hawksworth 1990).

Fuel persistence depends on site, climate, and mortality agent. Most of the literature on decomposition (Harmon et al. 19xx, Maser et al. 19xx) comes from west of the Cascades Crest and fails to differentiate among causal factors. However, Lundquist (1995) notes that trees killed by bark beetles were likely to have more sap rot than trees killed by root pathogens, which generally had more heart rot.

Root disease centers might function to break up fuel continuities and reduce fire spread because of their higher amounts of shrubs and herbs as noted by van der Kamp (1991).

Conclusion

Small-scale disturbance and tree mortality associated with native pathogens and insects is an on-going forest process in inland Western forests. Trees killed by pathogens and insects contribute woody material to forest ecosystems, material that is important for ecological functioning of these forests (Harmon.....xxxx several others). Besides contributing to wildlife habitat, soil formation, and nutrient cycling, dead wood also functions as fuel for the fires that are an inherent disturbance agent in inland Western forests.

In general, mortality reflected as the number of stems per ha is perhaps highest in forests that in the self-thinning (stem-exclusion / competition) phase of

development, with the pattern of this mortality relatively uniform. As forests develop into the gap-producing (understory reinitiation) phase, mortality levels probably decrease if reported as stems per ha, even though the basal area and volume of this mortality may increase. This gap producing mortality is less uniform, but may be locally high, especially where multiple agents are interacting. There is also a potential for fuel ladders to develop as forests become more uneven-aged. Mortality in very old forests may be high or low, depending on such things as species composition and species longevity. Forests approaching the upper age limit for the dominant species will experience elevated mortality. Initiating stands may experience high mortality although small-scale disturbances in very young stands may not contribute greatly to fuel loads; however, the initiating disturbance may have substantially elevated fuels in some young stands.

Matrix showing relative expected mortality levels for various pathogens by stand development phase (John - Do you think this is a good idea, and if so, perhaps you can improve on my concept).

	Initiating	Competition	Gap	Old
Armillaria	M-H	M	L	L-M
Annosus			M-H	M-H
Phelinus			M-H	M-H
DM - PP	M-H	M	L-M	L-M
DM - DF	M-H	M	H	H
DM - LP	M-H	H	L-M	L-M
DM - WL	L-M	L	M-H	H
DM - TF WH				
Bark beetles	L	L-M	L-H	M-H
Defoliators	L	L	L-M	M-H

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